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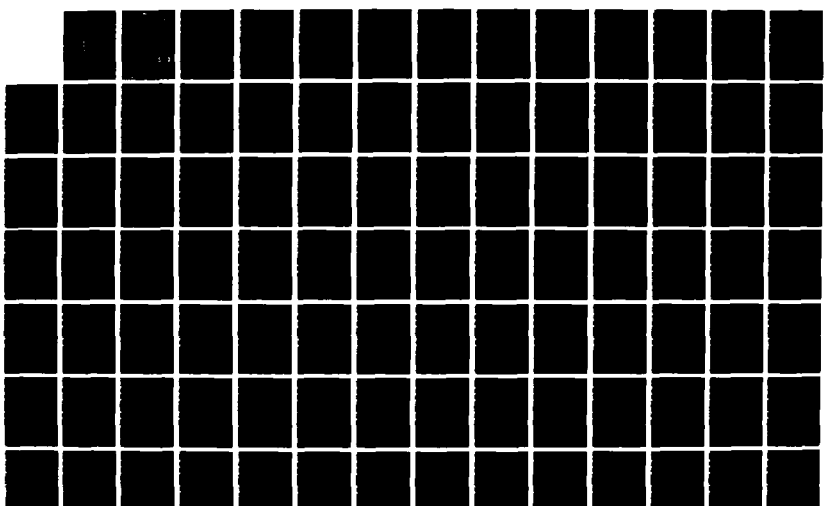

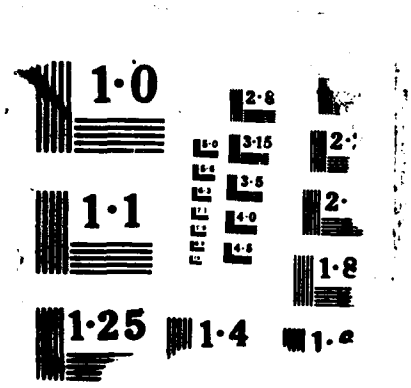
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Hypothermia has played a major role in the medical statistics from many armies deploying in a cold environment. The loss of body heat, either from the decreased heat production, increased heat loss, or alterations in thermoregulation are important in the epidemiology of this injury. Cold appears to protect various portions of the body from the decreasing cardiovascular capability of delivering nutrients and oxygen. Recognition of these patients as they present to medical facilities and recognizing they are treatable even when they are severely hypothermic is the key to survival. Emergency Department of Management involves fluid resuscitation, respiratory support, active core rewarming and management of electrolytes. Cardiovascular support is critical to long-term management and a successful outcome. Prevention is the key to indiviuals exposed to a high threat environment.

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Accidental Hypothermia

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Outline

Cold modalities, while used therapeutically for millenia, were not scientifically evaluated until the eighteenth century. Hemostatic, analgesic, and therapeutic effects of cold on a variety of conditions were well known. While fever remained the usual disorder of thermoregulation, accidental hypothermia was common, and its treatment controversial. The Bible cites truncal rewarming of King David by a damsel. Various other remedies were mentioned by Hippocrates, Aristotle, and Galen.[93,387]

Cold has turned the tide in many battles.[525] In addition to frostbite, the influence of hypothermia on military campaigns is evident throughout history. Xenophon experienced cold casualties in Persia around 400 B.C.[161] Alexander the Great was rendered comatose from hypothermia, as were many of the Roman legionnaires traversing the Alps. Hannibal lost approximately 20,000 of his 46,000 troops in 218 B.C. crossing the Pyrenees Alps in northern Italy.[22]

Barron Larrey, Napoleon's chief surgeon, returned to France in 1812 with 350 healthy soldiers from the initial 12,000 in the Twelfth Division. Descriptions of the mental and physical effects of the cold during the approach and retreat from the gates of Moscow included observations that cold victims placed closest to the campfire died.[41,268]

Washington's troops were battered by the winter of 1777-78, with nearly ten percent "left to perish by winter cold". In the trench warfare of the Crimean War (1845-1855), more than 1,000 French soldiers succumbed to the cold. The lessons learned at Crimea sixty years earlier were soon forgotten. Cold-related casualties reported during World War I included 115,000 British, 80,000 French, and 38,000 Italian troops. In World War II, the Germans suffered 100,000 and the U.S. 90,000 cold injuries. The high

mortality in downed German pilots in the North Atlantic and in U-boat crews led to the inhumane experiments at Dachau. Fully 10% of the total U.S. fatalities in Korea were cold related. At Goose Green in the Falklands War of 1982, the temperature dropped at night to -4°C and hypothermia, frostbite, and trench foot were common.[12,161]

Numerous cold-related tragedies have occurred in civilians, skiers, hunters, sailors, climbers, swimmers, and the indigent.[201,466,56,421,245,324,242,321,243] Civilian arctic and mountain expeditions have also been foiled. The toll on those challenging the environment has included those on Mt. McKinley, Mt. Everest, and most recently, Mt. Hood.

Epidemiology

The wide scope of hypothermia-related deaths warrants public health concern. The incidence of hypothermia in the U.S. is not clear. "Primary" hypothermia deaths are considered "violent", and are classified as either accidental, homicidal, or suicidal. Deaths from "secondary" hypothermia are usually considered natural complications of systemic disorders including trauma, carcinoma, and sepsis.[562] Death certificate data more accurately quantify primary hypothermia. Underreporting of cases, common in secondary hypothermia, can be caused by delays between hospital admission and death.[442] One report in the U.S. discovered only 19% of hypothermic victims survived to reach a hospital.[137]

In the first survey of a North American population, there was no evidence that low morning basal temperature is common at room temperature in a geriatric group.[247] In another epidemiological study in the U.S., a low morning body temperature was also uncommon in the elderly found

indoors.[414] In contrast, in a United Kingdom home survey, 11.4% of the elderly were hypothermic.[158] The accuracy of those urine temperatures has been questioned. A hospital based British study diagnosed hypothermia in 3.6% of admitted patients over 65 years of age.[176] The incidence of chronic hypothermia in the elderly is not decreasing, and in some populations fatalities were increased five times in those over 75 years of age.[415,416]

Hypothermia is geographically widespread during all seasons in the U.S. and abroad.[276,415,329,538,100,539,117,150,413] In a multicenter survey of 428 cases of civilian accidental hypothermia, 69 presented in Florida.[97] Urban settings account for the majority of cases in the U.S.

Hypothermia is both a symptom and clinical disease entity. Sufficient heat cannot be generated to maintain homeostasis. When the core temperature drops below 30°C, the patient becomes poikilothermic and cools to the ambient temperature.

Accidental hypothermia is an unintentional decrease in core temperature without preoptic and anterior hypothalamic nuclei disease. Several clinical classifications have been proposed. The most practical division includes healthy patients with simple exposure, those with specific diseases producing hypothermia, and those with predisposing conditions. Other divisions include immersion versus nonimmersion and acute versus chronic etiologies.[303,423,345]

Age extremes, state of health and nutrition, type of exposure, and intoxicant or medication ingestion jeopardize thermostability by decreasing heat production or increasing heat loss. The compensatory physiological responses to this challenge of heat loss via evaporation, radiation, conduction, and convection may fail.[303,423,546] Resultant mortality

ranges from 0% to well over 50% in clinical series, depending on risk factors.[329,538,539,303,93]

Safe experimental investigations of hypothermia in human volunteers terminate cooling at 35°C. This precludes study of some of the significant pathophysiology seen in moderate or severe hypothermia. Limitations also exist in animal studies, the results of which require variable degrees of extrapolation. Large differences exist in cardiovascular responses and peripheral musculature, particularly in non-porcine models. Clinical treatment recommendations must be predicated on the degree and duration of hypothermia and the type of predisposing factors.[195,196,68,452]

Normal Physiology of Temperature Regulation

Homeotherms juggle the dynamic equilibrium between heat production and heat loss. The normal diurnal variation in humans is only 1°F.[167] Since physiologic changes occurring in man are modified by predisposing or contributory factors, normal responses to severe temperature depression require extrapolation.[360,240,49]

Basal heat production is usually 40-60 Kcal/M^2 body surface area per hour. Food ingestion, fever, activity, and cold stress increase it.[168] Although discomfort from cold can be reduced over time, significant physiologic adaptation similar to that seen with heat stress does not appear to exist in humans. Normal thermoregulation in vertebrates involves transmission of cold perception to the hypothalamic neurons via the lateral spinothalamic tracts and the thalamus. (Figure 1)

Thermosensitive end-organs exist in the skin and possibly elsewhere.[482] Temperature sensitive hypothalamic neurons react directly to alterations in blood temperature.[38] Relevant hypothalamic nuclei

determine the temperature set-point. The direction and magnitude of compensatory responses are affected by physiological and biochemical factors. Multiple thermoregulatory neurotransmitters, prostaglandins, ions, and peptide neuromodulators alter these neuronal firing rates.[285,233] Dopaminergic and serotoninergic neurons appear pivotal.[353]

An integrated cascade of events follows perception of an acute cold stress. Preshivering muscle tone is increased.[402] Heat is generated by involuntary muscle contraction in the form of shivering, which is modulated by the posterior hypothalamus. Shivering thermogenesis can increase metabolic rate 2-5 times, until glycogen depletion and exhaustion develop.[222]

Shivering can begin and end within a few minutes after cold exposure. Oxygen consumption increases up to 3.7 times within 15 minutes.[222] The frequency of the shiver tremor is 8 - 11 Hz.

Pozos has demonstrated that thermal (cold) - induced shivering involves striking synchronization of differing muscle groups, in contrast to totally unsynchronized postoperative "shivering". (Figure 2). Since cold-induced vasodilation (CIVD) of digits is also synchronized at three to five per hour, it appears that shivering and CIVD are controlled by two separate neural oscillators.

Nonshivering thermogenesis results from diffuse metabolic activities. Thyroid hormone, adrenocorticoids, insulin, and catecholamines are involved. Cold activates the release of thyrotropin-releasing hormone from the hypothalamus. Subsequently the anterior pituitary produces thyroid-stimulating hormone, and the thyroid releases thyroxine.

In addition to endocrinologic reactions, a more rapid response is initiated by the autonomic nervous system.[167] Activation or suppression

of the sympathetic nervous system is seen with cold-induced release of norepinephrine.[229] Other homeostatic mechanisms may exist in humans. For example, primates are sensitive to ambient acceleration. Colonic temperature falls 2°C within minutes when squirrel monkeys are centrifuged.[166]

Adaptive behavioral responses affect the five normal mechanisms of heat loss. The amount lost varies with the temperature gradient between the exposed body surface and the environment.[469,35,86] Children cool most rapidly because of the elevated body surface area-to-mass ratios, and females generally cool more slowly than males.[243,50,399]

Radiation accounts for 55-65% of the usual heat loss. Radiative losses are least when the body is curled up and insulated, and determined by the amount of cutaneous blood flow and insulation. There is close correlation between cooling rates and subcutaneous fat thickness. Conduction and convection normally contribute about 15% to heat loss. Conductive losses are major in immersion hypothermia, and may increase five times in wet clothing and 25 times in water.[277] Shivering increases convective losses, as does swimming.[58,206,322,205,386]

The 20-30% heat loss from evaporation and respiration is markedly affected by the relative humidity and ambient temperature. Greatest losses develop in dry, cold, and windy climatic conditions.[303,399] Diaphoresis increases evaporative losses.

Normal physiological processes are activated or deactivated at each level of hypothermia. Between 35°C and 32.2°C, thermogenesis is predominantly from shivering and endocrinologic mechanisms which are coupled with vasoconstriction. Dropping from 32.2°C down to 24°C, vasoconstriction but not shivering is preserved, and metabolic heat

production gradually declines. Heat conservation mechanisms fail below 24°C.[303,22,203,499,33,220]

The physiologic characteristics of the three zones of hypothermia are represented on Table I.

Pathophysiology

CNS

Hypothermia depresses the central nervous system.[266,380] There is a linear decrease in cerebral metabolism of 6-7% per degree C from 35 to 25°C. The electroencephalogram is abnormal below 33.5°C, and becomes silent at 19-20°C.[152,129] Visual evoked potentials, an objective measure of cerebral function, become smaller as the temperature drops.[436] Temperature dependent enzyme systems present in the brain do not function at cold temperatures well tolerated by the kidneys.[41]

Cerebrovascular autoregulation is intact until the temperature drops below 25°C. Although vascular resistance is increased, there is a disproportionate redistribution of blood flow to the brain. In a canine study, blood flow recovered quickly to control levels in the brain, muscle, kidney, and myocardium after rewarming. Flow deficits persisted two hours post rewarming in the pulmonary, digestive, and endocrine systems.[16,537]

Cardiovascular

Cardiovascular responses in hypothermia are well described. Following an early tachycardia, progressive bradycardia results in a 50% decrease in heart rate at 28°C.[35] Since bradycardia is caused by decreased spontaneous depolarization of pacemaker cells, it is refractory to atropine.[403]

In hypothermic bradycardia, unlike normothermia, systole is prolonged greater than diastole. The conduction system is more sensitive to cold than

the myocardium, so the cardiac cycle is lengthened. Cold-induced fluxes in pH, oxygen, electrolytes, and nutrients also alter conduction. First the PR, then the QRS, and most characteristically the QT_c intervals are prolonged. Invisible preshivering muscle tone can artifactually obscure P waves. The ST segment and T wave abnormalities are inconsistent.[516,317]

The J wave (Osborn wave; hypothermic hump-Figure 3) was first described by Tomaszewski in 1938.[512] Present at the junction of the QRS complex and ST segment, it is not prognostic, but is potentially diagnostic.[381,433,250,447,553,178] J waves can be observed at any time below 32.2°C, and are most frequently present in leads II and V_6. When the core temperature falls below 25°C, they are most commonly found in the precordial leads (especially V_3 or V_4). The size of the J waves also increases with temperature depression, but is unrelated to arterial pH.[375,178] The J waves are usually upright in aV_L, aV_F, and the left precordial leads.

The cause of the J deflections is not clear, but may represent hypothermic ion fluxes resulting in delayed depolarization or early repolarization of the left ventricle. Another possibility is a hypothalamic or neurogenic factor. J waves are not pathognomonic of hypothermia, and are seen associated with central nervous system lesions, focal cardiac ischemia, in young normal patients, and in sepsis.[107,509]

Below 32.2°C, all atrial and ventricular dysrhythmias are commonly encountered.[119,136,77,509] Reentrant dysrhythmias are caused by decreased conduction velocity and absolute refractory periods in the setting of an increased myocardial conduction time. In one study between 25 and 20°C, myocardial conduction time was 400% of normal, while the absolute

refractory period only rose 228%. Independent electrical foci are another mechanism precipitating dysrhythmias.[88]

There are multiple explanations for ventricular fibrillation and asystole.[195,22] These rhythms occur spontaneously below 25°C. The fibrillation threshold is decreased, as is the transmembrane resting potential. An independent focus or re-entrant phenomenon develops. Since the heart is cold, a conduction delay is facilitated by the large dispersion of repolarization.[263] In hypothermia, the action potential is also prolonged.[8] This increased temporal dispersion of the recovery of excitability is linked to ventricular fibrillation.

Some reviews have implied asystole is the more common presenting rhythm, while ventricular fibrillation is iatrogenic.[22,479,145] This has not been documented in a multicenter survey.[97] Potential etiologies of both rhythms include therapeutic manipulations, tissue hypoxia, acid-base fluxes, autonomic dysfunction, and coronary vasoconstriction with increased blood viscosity.[537,495,523,348,291,496]

Hypothermia progressively decreases mean arterial pressure and cardiac index. The cardiac output drops to 45% of normal at 25°C. Systemic arterial resistance increased in a group of hemodynamically monitored patients.[194]

Core temperature "afterdrop" refers to the continual decline in a hypothermic patient's temperature after removal from the cold. The pathophysiology and clinical implications involved remain controversial.[489,423,195,196,202,543,197,175,227,101] There are two apparent processes that contribute to afterdrop. The first is simple temperature equilibration between a core which is warmer than the periphery. This explains the afterdrop that has been demonstrated on

isolated legs of beef and bags of gelatin.[531] Circulatory changes account for the other. Counter-current cooling of the blood perfusing cold tissues results in a core temperature decline until the temperature gradient is eliminated. This is particularly common during active external rewarming, where peripheral vasoconstriction and arteriovenous shunting is reversed.

The incidence of afterdrop has varied widely in clinical experiments and surgically induced hypothermia.[423,195,196,313,202,543,197,84,175,227,101,443] Hayward measured his own esophageal, rectal, tympanic, and cardiac temperature via flotation tip catheter after cooling in 10°C water.[204] On three different days, rewarming was attempted via shivering, heated humidified inhalation, and warm bath immersion. Mean arterial pressure fell 30% and peripheral vascular resistance fell 50% coincident with a 0.3°C afterdrop during warm bath immersion, supporting the circulatory hypothesis. Another study of peripheral blood flow during rewarming from mild hypothermia in humans suggests that only minimal skin blood flow changes contribute to afterdrop.[443] Harnett precipitated the largest afterdrops when subjects were rewarmed with plumbed garments and heating pads.[197]

Core temperature afterdrop appears to result from both circulatory changes and simple equilibration of temperatures. It is only clinically important in the treatment of patients with a large temperature gradient between the periphery and core.

For example, hypothermic patients experience major afterdrops when frostbitten extremities are thawed prematurely.

Respiratory

Respiratory rate stimulation is followed by progressive depression of respiratory minute volume as metabolism is depressed.[252] In severe

hypothermia, CO_2 retention and respiratory acidosis indicate altered responses to normothermic respiratory stimuli. Carbon dioxide production drops 50% for each 8°C fall in temperature.[303] Other pathophysiologic factors include decreased cilial motility, increased viscosity and amount of secretions, and noncardiogenic pulmonary edema.[70,377,481]

Renal

Renal blood flow is depressed by 50% at 27-30°C, which decreases glomerular filtration rate. Paradoxically, there is an initial large diuresis of dilute "glomerular filtrate".[448,449] This diuresis appears to be a response to the initial relative central hypervolemia induced by vasoconstrictive overload of capacitance vessels. Nitrogenous wastes are not cleared. There may be antidiuretic hormone inhibition and decreased renal tubular function.[449,211,85,236]

Progressive hemoconcentration occurs during cold diuresis. Blood volume decreases. Experimentally, cold water immersion also increases urinary output 3.5 times, and the presence of ethanol doubles that diuresis.[90]

Predisposing Factors

Factors predisposing to core temperature depression are those that decrease heat production, increase heat loss, or impair thermoregulation.[303,93]

Decreased Heat Production

Thermogenesis is often decreased at the age extremes. Neuromuscular inefficiency impairs shivering in conjunction with decreased activity and poor cold adaptation. Neonates have a large surface area-to-mass ratio, a relatively deficient subcutaneous tissue layer, and no behavioral defense mechanisms. Unadapted infants thermoregulate with initial vasoconstriction

and doubling of the metabolic rate. Adapted infants older than five days of age can increase lipolysis immediately, and burn oxidative brown adipose tissue.[390,189,428]

Emergent deliveries and resuscitations are responsible for most acute neonatal hypothermia. Lethargy, a weak cry, and failure to thrive are more common in subacute hypothermia. Many infants have paradoxical "rosy cheeks". After the first few days of life, hypothermia frequently indicates septicemia and carries a high mortality rate.[134,92,135,133] Low weight and malnutrition were common findings in a series of 56 hypothermic infants aged 4 to 113 days.[477] Hypothermia has been observed in the shaken baby syndrome.[298]

Aging progressively diminishes homeostatic capabilities. While most elderly have normal thermoregulation, they are prone to develop conditions that affect heat conservation.[20,50,414] The elderly are physiologically less adept at increasing heat production and the respiratory quotient, which is the ratio of the volume of carbon dioxide produced to the volume of oxygen consumed per unit of time.[303,424,176] Impaired thermal perception leads to poor adaptive behavior.[157,446,528] Decreased resting peripheral blood flow has been observed.[117,176,22,83,308] Metabolic studies demonstrate that lipolysis occurs in severely hypothermic elderly in preference to glucose consumption.[490] Spontaneous hypothermia has been seen in apparently healthy elderly patients.[308]

Endocrinologic failure, which includes hypopituitarism, hypoadrenalism, and myxedema, is another cause of decreased heat production.[170] Congenital adrenal hyperplasia with mineralocorticoid insufficiency is more common in cold climates. This may be an adaptive response to prolonged exposure to cold, since the "normal" cold diuresis is

reduced in these patients.[299] Hypothyroidism is often occult, with no available history of cold intolerance, dry skin, lassitude, or arthralgias. The degree of temperature depression correlates directly with mortality.[155] Eighty percent of those in myxedema coma are hypothermic, and it is several times more common in females.[123,1]

The result of insufficient fuel ranges from hypoglycemia to marasmus to kwashiorkor. Central neuroglycopenia distorts hypothalamic function.[491] In one series of predominantly alcoholic hypothermics, 41% were hypoglycemic.[151] Malnutrition decreases subcutaneous fat, and directly alters thermoregulation. In a series of 744 elderly females with femoral neck fractures, poor nutrition predisposed to hypothermia and attendant clumsiness.[25] Kwashiorkor is less often associated with hypothermia than is marasmus, because of the insulating (hypoproteinemic) edema.[438]

Hypothermia is also a greater threat than hyperthermia in marathon races in cool climates. Runners slowing from fatigue or injury late in a race were at serious risk of being hypothermic in a study of 62 runners.[320]

Increased Heat Loss

Healthy skin shields, sweats, and is a functional radiator. Poorly acclimated individuals suffer high diaphoretic, convective and evaporative heat losses during cold exposure. Any dermatologic malfunction increases heat loss. Erythrodermas include psoriasis, eczema, and exfoliative dermatitis.[422,261,184] Hypothermia with hypernatremic dehydration is seen in congenital lamellar ichthyosis.[169]

Burns, and overzealous burn treatment, are additional etiologies of heat loss.[287] Iatrogenic causes include massive cold intravenous

infusions and overcooling of heatstroke victims. Environmental immersion exposure is discussed in Chapter X, "Cold-Water Immersion".

There are multiple pharmacologic and toxicologic agents which increase heat loss and impair thermoregulation. The effects of ethanol are complex.[233,302] Ethanol interacts with every studied putative or proven thermoregulatory neurotransmitter, including serotonin and dopamine. Although ingestion leads to a feeling of warmth and perhaps visible flushing, it is the major urban cause of hypothermia.[4,511,147,219,97,329,538,213] In one series of 51 fatal cases of accidental hypothermia, two-thirds of the victims were under the influence of alcohol and half were considered "habitual drunkards".[4]

Ethanol increases the risk of being exposed to the environment by modifying protective adaptive behavior. Paradoxical undressing, the removal of clothing in response to a cold stress, is common.[4] Ethanol is a poikilothermic agent that directly impairs thermoregulation at high or low temperature. Body temperature is lowered from cutaneous vasodilatation and impaired shivering thermogenesis. Chronic ethanol ingestion damages the mammillary bodies and the posterior hypothalamus, which modulates shivering thermogenesis.[233,402]

Neurophysiological effects of ethanol are modified by the duration and intensity of exercise, food consumption, and the applied cold stress. Aging increases sensitivity to the hypothermic actions of ethanol in primate experiments.[349] Chronic ingestion yields tolerance to its hypothermic effects, and at times a rebound hyperthermia is seen during withdrawal. Conditions commonly associated with ethanol ingestion which adversely affect heat balance include immobility and hypoglycemia. Inhibition of hepatic gluconeogenesis coexists with malnutrition.[329,538,539,302]

Intravenous thiamine is diagnostic and therapeutic for Wernicke's encephalopathy.[193,241,112,393,301,425] The acute triad of global confusion, opthalmoplegia, and truncal ataxia is often masked, and temperature depression may persist for weeks.[193] Hypothermic alcoholic ketoacidosis has also been reported.[15]

Impaired Thermoregulation

Thermoregulation impairment can be categorized as central, peripheral, metabolic, pharmacologic, or toxicologic. Central conditions directly affect hypothalamic function and frequently mediate vasodilatation. Traumatic lesions include skull fractures, especially basilar, and intracerebral hemorrhages, most commonly chronic subdural hematomas. Pathologic lesions commonly associated with hypothermia include neoplasms, congenital anomalies, and Parkinson's disease.[159,326,69] Cerebellar lesions produce inefficient choreiform shivering. Cerebrovascular accidents result in hypothalamic dysfunction.

During Hodgkin's disease, hypothermia is seen exclusively in previously febrile patients with advanced disease independent of cell type.[54] This appears to be a disease-associated functional disorder of thermoregulation, similar to hypothermia associated with anorexia nervosa. Of interest, centrally induced hypothermia has been antagonized completely with thyrotropin releasing hormone.[210]

Peripheral thermoregulation fails after acute spinal cord transection.[18,7] Victims are functionally poikilothermic when peripheral vasoconstriction is extinguished.[397] Other peripheral causes include neuropathies and diabetes. Hypothermia is more common in elderly diabetics than in the general population, even after exclusion of diabetic metabolic emergencies.[358] The common denominator in metabolic derangements may be

the abnormal plasma osmolality interfering with hypothalamic function. Additional causes include hypoglycemia, diabetic ketoacidosis, and uremia.[498,224] The pH was 6.67 in one hypothermic survivor with lactic acidosis.[351]

Numerous medications and toxins in therapeutic or toxic doses impair centrally mediated thermoregulation and vasoconstriction. In a single series of 103 critical overdoses, 27 were hypothermic.[232] Offenders include general anesthetics, barbiturates, benzodiazepines, phenothiazines, and the cyclic antidepressants. Reduction of core temperature may be a prodrome of lithium poisoning.[153] Organophosphates, narcotics, glutethimide, clonidine, fluphenazine, bethanechol, atropine, acetaminophen, and carbon monoxide have all been reported to cause hypothermia.[144,265,181,315,281,267,115,303,367,391,20]

A variety of clinical entities are associated with hypothermia. Traumatic conditions causing hypotension and hypovolemia jeopardize thermostability.[389,25,239,29,3,434,12] A decrease in skin and core temperature without compensatory shivering thermogenesis has been reported in a group of major trauma patients as defined by the Injury Severity Score.[286] Recognition of hypothermia is often obscured by obvious hemorrhage and injuries. Traumatic neurologic deficits including areflexia and paresis can be misattributed to hypothermia.[18,397,7]

Recurrent and episodic hypothermia is widely reported.[472] Recurrent is more common, and usually is secondary to ethanol abuse. One patient has survived 12 episodes.[330] Severe recurrent presentations have also been caused by self-poisoning and anorexia nervosa.[474,115]

Despite significant overlap, there are two groupings of patients with "episodic hypothermia".[508] One group is hypothermic for hours.

Diaphoretic episodes precede the temperature decline. Included in this group are patients with hypothalamic lesions and agenesis of the corpus callosum, termed the Shapiro Syndrome.[461,396] Hypothermia with agenesis of the corpus callosum has also been associated with hypercalcemia and status epilepticus.[230] Since experimental sectioning of the corpus callosum does not induce hypothermia, associated lesions including lipomas probably cause the thermoinstability.[280,493,440]

The first group with brief episodic hypothermia also includes those with spontaneous periodic hypothermia.[160] This condition may represent a diencephalic autonomic seizure disorder.[118,337] Florid psychiatric symptoms mask the intermittent hypothermic episodes. Idiopathic periodic hypothermia has also been noted in a patient with occult syringomyelia and bizarre behavior.[180]

The second category of patients with episodic hypothermia remain cold for days to weeks. These patients have more seizure disorders, and the central thermostat is set low. Patients with intermittent hypothermia usually demonstrate characteristics from both groups.[303,330,508]

Multiple infestations and infections may both elevate and depress temperature, including septicemia, pneumonia, peritonitis, meningitis, and ncephalitis, bacterial endocarditis, typhoid, miliary tuberculosis, syphilis, brucellosis, and trypanosomiasis.[53,279,303] Other diseases that produce secondary hypothermia include carcinomatosis, pancreatitis, cerebrovascular and cardiopulmonary disorders.

Hypothermia resulting from low cardiac output after myocardial infarction was reversed in one patient with intra-aortic balloon counter pulsation.[109] Magnesium sulfate infusion during preterm labor has caused hypothermia with fetal and maternal bradycardia.[429] Additional

etiologies include vascular insufficiency, giant cell arteritis, sickle cell anemia, Paget's disease, and sarcoidosis.[303]

Presentation

Historical circumstances frequently suggest the presence of hypothermia. When exposure is obvious, such as in avalanche victims, the diagnosis of hypothermia is simple. Subtle presentations predominate in urban settings, with vague symptoms including hunger, nausea, chills, and dizziness. Predisposing underlying illnesses or ethanol ingestion are commonly present. Major trauma, immersion, overdose, and cerebrovascular accidents are further presentations.[288,289,264,507]

Usually the constellation of physical findings suggests the diagnosis. During the HEENT examination, abnormal findings include decreased corneal reflexes, mydriasis, strabismus, flushing, erythropsia, facial edema, rhinorrhea,and epistaxis. Cardiovascular findings include bradydysrhythmias and hypotension after the initial tachycardia. Heart sounds may be muffled

Tachypnea, an early respiratory finding, may be followed by progressive hypoventilation accompanied by bronchorrhea and adventitious sounds. The gastrointestinal tract is depressed. Gastric dilatation is particularly common in neonates and in myxedematous adults. Abdominal distention or rigidity, ileus, obstipation, and poor rectal tone are frequently present. Genitourinary output ranges from anuria to polyuria. The incidence of testicular torsion increases because of cremasteric contractions.

Diffuse neurological abnormalities are usually present, but vary widely. Some patients converse at 25°C, and are normoreflexic. The presence of ataxia and dysarthria may mimic a cerebrovascular accident.[176] There is a progressive decrease in the level of

consciousness proportionate to the degree of hypothermia. Speed of reasoning and memory registration is impaired.[81] Amnesia, antinociception, anesthesia, or hypesthesia develop. Cranial nerve signs are present following bulbar damage from central pontine myelinolysis.[504] Extraocular muscle movement abnormalities, similar to extensor plantar responses, do not directly correlate with the degree of hypothermia.[148]

Hyperreflexia predominates from 35°C to 32.2°C, and is followed by hyporeflexia. The plantar response remains flexor until 26°C, when areflexia develops. The knee jerk is usually the last reflex to disappear, and is the first to reappear during rewarming.[303] From 30°C to 26°C, both the contraction and relaxation phases of the reflexes are prolonged equally. However in myxedema, the relaxation phase of the ankle reflex is prolonged greater than the contraction phase.[306] When males are thoroughly chilled, cremasteric reflexes are absent. Spinal cord and other CNS lesions may be obscured by depressive neurologic changes normally accompanying hypothermia.[148,18]

Hypothermic psychiatric presentations and suicide attempts are commonly misdiagnosed. Pre-existent psychiatric disorders blossom in some individuals who are adjusted to temperature climates.[357,356] Mental status alterations include anxiety, impaired judgement, perseveration, neurosis, and psychosis.[441] Leaders of expeditions become moody, apathetic, and uncooperative risk takers.[406] Elderly patients often withdraw in confusion, become silent, and display lassitude and poor judg ment. A peculiar flat affect is common. Psychomotor impairment resembles organic brain syndrome.[81,506]

Appropriate adaptive behavior is often lacking.[82] An extreme example, "paradoxical undressing", is widely reported. Clothes are removed

in a preterminal effort to address thermoregulatory collapse. Patients have been mistaken as sexual assault victims.[6,532] Paradoxical undressing is also seen in children.[471]

Musculoskeletal posturing can extend to pseudo rigor mortis. Pre-shivering muscle tone is increased. Muscular rigidity, paravertebral spasm, and opisthotonos may be present. Compartment syndromes may result from conditions causing prolonged immobility.[383,411]

Dermatologic presentations include erytherma, pallor, edema, and scleredema. Cold urticaria, frostnip, frostbite, and gangrene also suggest the diagnosis.[231] Pernio may be seen in association with chronic myelomonocytic leukemia.[248]

LABORATORY

Acid-Base Balance

Should arterial blood gas parameters be temperature-corrected? The bicarbonate content and oxyhemoglobin saturation do not appreciably change with temperature. It appears pointless to correct the pressures or intensities (PO_2, PCO_2, pH) that do change.[23,192,486]

Correction was initially suggested to aid in the clinician's interpretation of the pathophysiology involved in hypothermic arterial oxygenation and acid-base balance.[432,430,456] Correction creates problems. If a pH electrode could be used at the patient's current core temperature, an uncorrected but exact pH value would be obtained. However, arterial blood samples are always warmed to 37°C before electrode measurements are obtained, and are not measured at the patient's subnormal temperature.

To correct for changes in temperature, Severinghaus mathematical corrections are subsequently applied.[456,457] In an airtight syringe with

a constant CO_2 content, the Δ pH/°C of blood in vitro is - 0.0147. Thus, one could correct the pH by adding 0.0147 pH units per degree C below 37°C. The PaO_2 drops 7.2%/°C and the $PaCO_2$ drops 4.4%/°C decrease in temperature.[61,249] The oxyhemoglobin dissociation curve (Figure 4) shifts to the left because of the decreased partial presure of dissolved gases. To interpret temperature-corrected values, the clinician must compare results with the "normal" values at that temperature. For example, a PCO_2 of 40 mm Hg and a pH of 7.4 at 37°C is the equivalent of a PCO_2 of 30 mm Hg and a pH of 7.50 at 30°C. A PO_2 of 120 mm Hg at 37°C corresponds to a PO_2 of 59 mm Hg at 27°C and does not indicate arterial oxyhemoglobin desaturation.[486]

On the other hand, to accurately interpret uncorrected ABG's as they are measured by electrodes at 37°C, one need only compare values with the well known normal values at 37°C. This simplifies comparison of results from serial samples during rewarming. If the pH is 7.4 and the PCO_2 is 40 mm Hg, there is normal alveolar ventilation and acid-base balance.

Oxygen saturation measured directly or calculated from the pH and PO_2 at 37°C accurately reflects oxygenation since it does not change with temperature. In contrast, if values are temperature-corrected they must be compared with the temperature-corrected "normal" values which are continuously changing during rewarming.

Acid-base balance and strategy in hypothermia differs from normothermia in many regards. After an initial respiratory alkalosis from hyperventilation, the more common underlying disturbance is a relative acidosis. Respiratory acidosis is mainly caused by direct respiratory depression. In addition, as the temperature decreases the solubility of

CO_2 in blood increases. Contributors to metabolic acidosis include impaired hepatic metabolism and acid excretion, lactate generation from shivering, and decreased tissue perfusion.[120] Reliable clinical prediction of acid-base status in accidental hypothermia is not possible. In one series of 135 cases, 30% were acidotic and 25% alkalotic.[329]

Circulatory changes also prevent adequate mobilization and delivery of organic acids to buffer systems. As in normothermia, mixed venous blood may best reflect acid-base status during resuscitation.[533] Despite flow changes, in a moderately hypothermic canine model a significant correlation persists between arterial and mixed venous pH.[338] The arterio-venous Δ pH was $\pm$ 0.03 to 0.04 pH units. This is coupled with the markedly impaired buffering capacity of cold blood. In normothermia, when the PCO_2 increases 10 mm Hg, there is a decrease in pH of 0.8 units. At 28°C, the decrease in pH doubles.

Optimal strategy to maintain acid-base homeostasis during treatment of accidental hypothermia is being challenged. The accepted earlier assumption was that 7.42 is the ideal "corrected" patient pH at all temperatures, and that therapy should be directed at maintenance of the corrected arterial pH at 7.42. The rationale for applying this 37°C criterion to cold tissues is being questioned.[544,545] A better intracellular pH reference may be electrochemical neutrality, where pH = pOH. Since the neutral point of water at 37°C is pH = 6.8, Rahn has hypothesized that this normal 0.6 unit pH offset in body fluids should be maintained at all temperatures.[409,410,419,455] Since the neutral pH rises with cooling, so should blood pH (Figure 5).

Depressed metabolism and CO_2 generation are a physiologic response to temperature depression. Each temperature has its associated metabolic

rate. Ventilation is intrinsically adjusted to maintain a net charge on the defended parameter, the peptide-linked histidine imidazole buffering system. Relative alkalinity of tissues makes physiologic sense. Intracellular electrochemical neutrality ensures optimal function of the enzyme systems and transport proteins at all temperatures.[23]

Rahn's hypothesis was motivated in part by his observation that Antarctic codfish survive far below the freezing point of water because of a glycoprotein antifreeze and function in an extremely alkalotic state. This same blood pH variation, that is a rise in pH with a decline in temperature, is seen in other cold blooded vertebrates and invertebrates.[409,410]

One homeostatic approach to maintain a steady pH is to keep bicarbonate content constant. This is achievable if total blood CO_2 content also does not change. Since CO_2 solubility increases with temperature depression, alveolar ventilation must increase to compensate by lowering the PCO_2. Poikilotherms exhibit this respiratory adaptation, and do not depress respiratory minute volume when cold. They maintain the total HCO_{3-} and CO_2 content at normothermic levels by decreasing PCO_2 while increasing pH. Hibernating mammals are more acidic, employing an acid-base strategy which suppresses metabolism.[352] At a cost of increased total HCO_{3-} and CO_2 content, they maintain a normothermic pH and PCO_2.

There are several experimental and clinical studies which support Rahn's hypothesis. In one study, a set of puppies with pH maintained at 7.4 had a 50% drop in cardiac performance after bypass.[26] The control group, left alkalotic, had normal cardiac indices and increased cerebral blood flow.[258,497,495] During systemic deep hypothermia in further canine studies, constraining the corrected pH to 7.4 caused myocardial

damage.[26] Relative alkalinitiy afforded myocardial protection. Other advantages include improved electrical stability of the heart. The fibrillation threshold of dogs markedly decreased when arterial pH was held at 7.40, but was unchanged with alkalosis. In contrast, maintaining the pH at 7.4 during hypothermia in a rat model did not effect cardiac work response.[468] This suggests the range of optimal extracellular pH is large in some species.[495]

During cardiopulmonary bypass, CO_2 is excreted and blood in the oxygenator does not reach equilibrium. One ventilatory regimen including 5% CO_2 was optimal in a study of induced hypothermia.[191] Using 1-2% CO_2 rather than 5% CO_2 in the oxygenator produced an uncorrected PCO_2 nearer 40 mm Hg and relative alkalosis in a study of 28 children on bypass with deep hypothermia.[319] Another advantage in adding a small fraction of CO_2 to the inspired mixture is the flattening and shifting of the oxyhemoglobin curve to the right (Figure 4).[405]

In a recent study of 181 cardiac bypass patients, 121 consecutive cases were "endothermically" managed with corrected normal pH and PCO_2 values.[260] Ventricular fibrillation occurred in 49 (40%). In the other 60 cases left "ecotothermically" alkalotic, only 12 patients (20%) developed spontaneous ventricular fibrillation.

These observations provide some evidence in support of Rahn's hypothesis that the advantage ectotherms obtain with a constant relative degree of alkalinity also applies to warm blooded endotherms during hypothermic conditions. Potentially deleterious effects of this alkalosis on other organ systems have yet to be identified. However, it is clear that maintaining the corrected pH at 7.4 and PCO_2 at 40 mm Hg during hypothermia depresses cerebral and coronary blood flow, cardiac output,

and increases the incidence of lactic acidosis and ventricular fibrillation. Correction of pH and PCO_2 in hypothermia is unnecessary and potentially deleterious.

HEMATOLOGIC

Hematologic evaluation is needed in all but mild exposure cases. The hematocrit will appear to increase from a decline in plasma volume and a 2% increase per 1°C fall in temperature.[235] The total red blood cell mass is low if an antecedent anemia, malnutrition, leukemia, uremia, or neoplasm is present.

The white blood cell count is frequently normal or low despite the presence of sepsis. Leukopenia does not imply absence of infection if the patient is at either age extreme, debilitated, intoxicated, myxedematous, or has secondary hypothermia.[464,35,279] The leukocyte count drops in hypothermia because of bone marrow depression and hepatic, splenic, and splanchnic sequestration.

Serum electrolytes must be continuously rechecked during warming. Data from experimental and clinical settings demonstrate no trends, and there are often no safe predictors of values.[195,22,146,97] Serum electrolytes fluctuate with temperature, duration of exposure, and rewarming technique. Membrane permeability and sodium-potassium pump efficiency change with temperature.[399]

Isolated temperature depression has no consistent effect on sodium and chloride levels until well below 25°C.[97,427] Plasma levels are affected by fluid shifts, prehydration, rehydration, and endocrine or gastrointestinal dysfunction.

The plasma potassium level is independent of temperature.[237,102,236,427] Hypokalemia may result from potassium

shifting into muscle, and not from kaliuresis.[36] The apparent discrepancy of a decreasing potassium level while pH is decreasing may be explained by greater intracellular than extracellular pH changes. Hypokalemia is more common in prolonged or chronically induced hypothermia.[256,374,19]

Prior diuretic therapy, alcoholism, diabetic ketoacidosis, hypopituitarism, and inappropriate antidiuretic hormone secretion exacerbate potassium deficiencies. Hypokalemic digitalis sensitivity can be masked by hypothermia. Gradual correction of hypokalemia during rewarming is required to optimize cardiac and gastrointestinal function.[423,146,325] Administration of bicarbonate, insulin, or calcium should be conservative.

Identification of hyperkalemia should suggest other causes of metabolic acidosis, rhabdomyolysis, or renal failure. Temperature depression will increase hyperkalemic cardiac toxicity. Diagnostic EKG changes are often obscured, and ventricular fibrillation occurs with serum potassium levels below 7 meq/L.

Cellular membrane transport inhibition decreases glucose utilization. In addition, insulin release and activity are reduced below 30°C. Target cells are insulin resistant. Hyperglycemia is common initially. Markedly elevated glucose levels often correlate with hyperamylasemia and increased cortisol secretion.[539,490,351,91,151,523,304,307,403,475]

Acute hypothermia also elevates serum glucose levels via catecholamine-induced glycogenolysis. Chronic exposure following exhaustion and glycogen depletion leads to hypoglycemia. Symptoms often mimic those attributed to hypothermia. Cold-induced renal glycosuria is common, and does not imply normo- or hyperglycemia. When hypoglycemia and central neuroglycopenia are present, correction will only improve the level of consciousness to that expected for the current core temperature.[151,163]

Hyperglycemia that persists during rewarming signals the potential for diabetic ketoacidosis or hemorrhagic pancreatitis. Insulin is ineffective until well above 30°C, and should be withheld to avoid iatrogenic hypoglycemia after rewarming.[163] Blood urea nitrogen (BUN) and creatinine are often elevated because of decreased nitrogenous waste clearance by the cold diuresis. Prior renal disease is a possibility. BUN is a poor reflector of volume status because of ongoing fluid shifts.[296,146]

Hypothermia has no consistent effect on magnesium or calcium levels. Severe hypophosphatemia was reported in one patient during treatment of profound hypothermia.[278] An intracellular phosphate shift is postulated since urinary excretion was minimal.

Numerous serum enzymes are elevated when diffuse ultracellular structural damage occurs in severe accidental hypothermia.[312,59,305] This is not seen in mild experimental hypothermia. CPK levels over 200,000 IU have been observed. Rhabdomyolysis is often present in these instances.[411]

The relationship between hypothermia and hyperamylasemia is poorly defined. It appears to correlate with the severity of temperature depression.[418,200,62] Pre-existent or hypothermic induced pancreatitis is common in up to 50% of the patients in some urban series.[307,304,119] Since abdominal examination is unreliable, an amylase level should be obtained except in minor cases.

Hyperamylasemia can correlate with mortality.[539] Ischemic pancreatitis is attributed to microcirculatory collapse in hypothermia.[156] Decreased pancreatic blood flow activates proteolytic enzymes.[418] Cholesterol and triglyceride levels are also often low.[490]

Hypothermia in the presence of hypothalamic astrocytomas and pancreatitis has been reported.[62,200]

A full clotting screen including platelet count and fibrinogen level is mandatory in all but mild cases of hypothermia. Thrombocytopenia usually reverses with rewarming, but adverse trends may signal impending problems. Physiologic hypercoagulability may exist in hypothermia, with a DIC-type syndrome.[309,437] Humans, unlike hibernating mammals, lack the vasomotor ability to ensure perfusion with alternative vasoconstriction and vasodilatation.[208] There is a higher incidence of thromboembolism. Postulated causes include thromboplastin release from cold tissue, simple circulatory collapse, and catecholamine and steroid release.[65]

Prolonged clotting times with leukopenia and thrombocytopenia are very significant at 20°C.[470,526] Cold-induced thrombocytopenia has been observed in 17 patients undergoing induced hypothermia. The average platelet count dropped from 184,000 to 37,000/ml^3 with maximal cooling.[464] The exact mechanism and site of this cold induced sequestration of platelets with neutrophils is unclear. Direct bone marrow suppression and splenic or hepatic sequestration have been proposed.[371,431,395] Thromboxane B2 production by platelets is temperature-dependent. Cooling of skin temperature in baboons produces reversible platelet dysfunction.[522,70] Thrombocytopenia may be a common but poorly recognized complication in the elderly and neonates.[122,80] A distinctive hypothermic hematologic picture has been reported to includethrombocytopenia, sideroblastic anemia, and erythroid hypoplasia.[180,371]

Hematologic abnormalities are common. The average temperature of 123 initially normothermic trauma patients who developed lethal coagulopathies

was 31.2°C.[239] In neonatal cold injury, thrombocytopenia was present in 6 of 7 infants. Since fibrin split product levels were normal, this was not felt to be a manifestation of DIC.

Cold hemagglutination results in either hemolysis or thrombosis.[108] The elevated viscosity of hypothermia is exacerbated with cryoglobulinemia. Cryofibrinogen is a cold-precipitated fibrinogen occasionally seen in conjunction with carcinoma, sepsis, and collagen vascular disease. Another mechanism that increases blood viscosity is the transient initial increase in platelet and red cell counts seen with mild surface cooling.[70] This could help to explain the increased mortality from coronary and cerebral thrombosis in winter.[246] The value of streptokinase and tissue plasminogen activator for hypothermic-induced thrombosis is unknown.

Pre-Hospital Management

Field treatment of hypothermia is the art of the possible.[56] Since cold, stiff, cyanotic patients with fixed dilated pupils have been reanimated, the treatment dictum for prehospital personnel remains "no one is dead until warm and dead".[125,182] Pre-hospital estimates regarding the potential reversibility of illness or injury should be conservative pending rewarming.

History obtained on the scene helps determine optimal treatment. A chronic subclinical course of hypothermia in an elderly indoor victim presents different challenges than an acute exposure or immersion episode. Any pertinent past medical history regarding cardiopulmonary, endocrinologic, or neurologic conditions is helpful.[66] The circumstances of discovery, duration of exposure, associated injuries or frostbite, Glasgow Coma Scale, and obvious predisposing conditions should be recorded.

Accurate field measurement of core temperature is often not practical. Indoors, truncal skin temperature may suggest the severity of hypothermia. Low-reading oral thermometers which record down to 25°C give an estimate of the temperature in cooperative patients who are not tachypneic. These thermometers are unreliable outdoors in cold ambient conditions.[420]

Prolonged field treatment should be avoided whenever possible, and the rescuer must prevent further heat loss. Passive external rewarming with dry insulating materials will minimize conductive, convective, evaporative, and radiative heat losses. Stimulants and oral heated fluids are not useful. Severely hypothermic patients should be handled gently and immobilized, and not allowed to exert themselves. Massage of cold extremities is contraindicated. Skin rubbing, like ethanol, suppresses shivering thermogenesis and increases cutaneous vasodilatation.

Prolonged field rewarming may be needed until conditions develop more favorably for land or preferably aeromedical evacuation, which prevents jostling of the victim.[11] Severe hypothermia must be presumed if a patient remains unresponsive following administration of 50% dextrose and naloxone. One must anticipate an irritable myocardium, hypovolemia, and a temperature gradient between the periphery and the core: the patient is in a "metabolic ice box".[332]

An intravenous fluid challenge with 250 - 500 cc heated 5% dextrose in normal saline should be given if available. During transport, a plastic intravenous container can be placed under the patient's back, providing warmth and infusion pressure.

Selection of options to stabilize the core temperature should be tailored to the severity of hypothermia and field circumstances. Gentle removal of wet clothing while the patient is prone may be essential to

limit heat loss and orthostasis. Passive external rewarming with insulation suffices for mild chronically induced hypothermia.[226] Active field rewarming techniques should be avoided unless heated humidified oxygen is available or if evacuation will be delayed.

Based on rewarming experiments, Harnett contends that the only safe active rewarming technique in the field for profound hypothermia is inhalation therapy.[197] It prevents respiratory heat loss, which represents a large percentage of heat production when the core temperature is below 32°C. Respiratory heat losses also vary with humidity, ambient atmospheric temperature, and the patient's respiratory minute volume (RMV).[292]

Lloyd initially recommended inhalation rewarming in 1971. His initial field device generated heat and moisture produced from a CO_2 and soda lime reaction.[290,292] The original closed-circuit prototype with oxygen tank weighed 8 kg. The current version weighs 3 kg including oxygen.[295] This unit consists of an oxygen cylinder, demand valve, two liter reservoir bag, soda lime and pediatric waters canister. An in-line thermometer measures mean air temperature at the face mask.

Hayward and Douwens' Uvic® Heat Treat® Systems (Thermo-Genesis Int. Inc.) are lightweight, portable first aid devices that deliver heated humidified air at all operating temperatures in the field. This system has a clear oronasal mask connected via a corrugated hose to a temperature control valve. (Figure 6) Steam generated via propane, camp-pot, or an electric system mixes with ambient air. The inhalant temperature is measured at the mask. Supplemental oxygen and ventilation can be provided with a bag adaption and the system can be modified to treat three patients.

The Hypothermia Oxygen Warmer treatment device (Bow/Parm, Inc.) is

another system capable of delivering heated humidified oxygen in the field. It also includes a Micro Temperature Computer to monitor oxygen and patient temperatures.

Although surface rewarming suppresses shivering, which impedes the rate of core rewarming, it may be required when the victim is isolated from medical care. Active external rewarming options include radiant heat, warmed objects placed on the patient, and body-to-body contact. Care must be exercised not to burn patients with hot objects, including commercially produced "hot packs". Experimentally, core temperature afterdrop was recorded with isolated upper truncal contact.[197] Total body contact rewarming may be hazardous. A hydraulic sarong or vest, in which heated water is circulated via hand pump, has been used.[17] Immersion rewarming is dangerous in the field because monitoring and resuscitation capabilities are limited.

Pre-hospital life support for hypothermia differs from that for normothermia in some regards (Figure 7). A patent airway and the presence of respirations must be established. The patient may appear apneic if there is significant depression of RMV. Overzealous assistance of ventilation can induce hypocapneic ventricular irritability.

Palpation of peripheral pulses is difficult in vasoconstricted and bradycardic patients. Apparent cardiovascular collapse may actually be depressed cardiac output, often sufficient to meet minimal metabolic demands. Prolonged palpation and auscultation of at least a minute for any spontaneous pulses may be necessary. Iatrogenic ventricular fibrillation can result from unindicated chest compressions.

If a cardiac monitor is available, defibrillation once with 2 wsec/kg up to 200 wsec is indicated for ventricular fibrillation or apparent asystole.[501,323] Unresponsive patients must be carefully assessed for a central pulse before they can be considered to have electromechanical dissociation. The lowest successful reestablishment of mechanical cardiac activity has been at 20°C.[99] Defibrillation attempts rarely succeed below 30°C.[303,291] If resuscitation in the field is unsuccessful, rewarming and CPR should be continued enroute to the emergency department. (See CPR section). A summary of prehospital care is to rescue, examine, insulate, and transport.[488]

ED Management

Hypothermia must be confirmed with core temperature measurements. The most common ED diagnostic errors result from incomplete vital signs. Asynchronous respirations should be averaged over several minutes. A Doppler may be required to locate a pulse, supported by continuous electrocardiographic monitoring. Advanced life support is initiated when necessary (Figure 8). The history obtained from a hypothermic patient should be considered unreliable.[297]

In some hospitals, adequate equipment for accurate core temperature measurement is unavailable.[465] Rectal measurements are most practical clinically, but may not reflect cardiac or brain temperatures.[127,548] Deep placement of the indwelling thermistor probe to 15 cm. is reliable unless placed into cold feces. The rectal temperature lags behind core temperature fluctuations, and is affected by lower extremity temperatures.[530,127,541,548,42]

Simultaneous esophageal temperature measurements may be helpful when airway protection is provided with endotracheal intubation. Since the upper

third of the esophagus is in proximity to the trachea, probe reliability is often poor during heated inhalation therapy.[543] The tympanic temperature most closely approximates hypothalamic temperature.[313,202,530] Tympanic measurements, not commonly done clinically, can be obtained with an infrared Diatek® device. The correct selection, accuracy, and use of urine temperature measuring devices is debated.[158,247,128] Urine temperature has been of mixed value in screening for hypothermia in elderly patients and is not recommended.[282]

After temperature measurement, all clothing should be gently removed or cut off with minimal patient manipulation, and the patient immediately insulated with dry blankets. A cardiac monitor is applied and intravenous catheters inserted as needed. Arterial catheter insertion has helped in the management of selected profoundly hypothermic patients. Central venous and pulmonary artery catheters have precipitated cardiac arrhythmias and should be reserved for complex cases.[385,341]

Laboratory evaluations except in some cases of mild hypothermia include a Dextrostix, blood sugar, arterial blood gases, complete blood count, electrolytes, BUN, creatinine, serum calcium, serum magnesium, serum amylase, prothrombin and partial thromboplastin times, platelet count, and fibrinogen level. A toxicologic screen should be considered if the level of consciousness does not correlate with the degree of hypothermia. Selective use of thyroid function studies, cardiac isoenzymes, and serum cortisol is indicated.

Cervical spine films in poorly responsive patients detect occult trauma. Chest radiographs may predict rewarming collapse when cardiomegaly and redistribution of vascularity are present. Bowel sounds are usually diminished or absent in severe cases.[303] Abdominal physical examination

is unreliable, and rectus muscle rigidity is frequently present. Abdominal films should be obtained and may reveal pneumoperitoneum, pancreatic calcifications, or hemoperitoneum. Small bowel dilatation is seen with cold-induced mesenteric vascular occlusion. Colonic dilatation is seen in conjunction with *myxedema coma*.

Nasogastric tube insertion should follow endotracheal intubation in moderate or severe hypothermia. Gastric dilatation and poor gastric intestinal motility are common. In-dwelling bladder catheters with urimeters are needed to monitor urinary output and cold diuresis.

Fluid Resuscitation

Most fluid shifts reverse with rewarming, and mild hypothermia only requires an IV lifeline. In more severe cases, volume shifts and elevated blood viscosity from the hemoconcentration, lowered temperature, increased vascular permeability, and low flow state mandate fluid resuscitation.[366] Blood viscosity increases 2% per degree C temperature drop, and hematocrits over 50% are seen. Low circulatory plasma volume is often coupled with elevated total plasma volume during rewarming.[195,22] Hemodilution is usually only seen during crystalloid resuscitation of hemorrhaging patients.

Dehydration is frequently present, with free-water depletion elevating serum sodium and osmolality. Normal physiologic cues for thirst are inactive. Since hypothermia produces natriuresis, saline depletion may be present. Further causes of low sodium include prior diuretic therapy and gastrointestinal losses. Pre-existent sodium excess is seen with congestive heart failure, cirrhosis, or nephrosis. In these cases, serum sodium and osmolality values are often normal. Rarely, serum sodium is low from

free-water excess. Other etiologies include myxedema, panhypopituitarism, and inappropriate antidiuretic hormone secretion.[93,303]

Most patients below 32.2°C should receive an initial fluid challenge with 250-500 cc of 5% dextrose in normal saline. Ringer's lactate solution should be avoided, since a cold liver cannot metabolize lactate. Any advantage of colloids over crystalloids is unclear. In one experiment, normal saline had minimal lasting effects and did not hasten cardiovascular recovery from hypothermia.[427] In another, 10% low molecular weight dextran solution increased plasma volume and decreased blood sludging.[114] Administer colloids only in patients not responding to crystalloids.

The safety of pneumatic anti-shock garments in hypothermia is not known. Their application presents several theoretical circulatory and limb hazards. Since the vasculature is already maximally vasoconstricted, provision of more peripheral vascular resistance by the garment should not be possible. Hypothermic patients are at high risk for extremity compartment syndromes and rhabdomyolysis. Do not use MAST trousers except for temporary stabilization of exsanguinating major pelvic fractures.

If near-drowning is the cause of hypothermia, routine crystalloid resuscitation is not warranted. Use of hypertonic saline for fresh water or hypotonic saline for salt water near-drownings is not justified. (See Chapter "Near Drowning")[39,111,199,198,335,244,37,467,450,9]

Standard clinical signs of fluid overload including rales, jugular venous distention, hepatojugular reflux and an S_3 gallop should be monitored. Persistent cardiovascular instability often reflects inadequate intravascular volume.[22,146,194,140] In these cases, properly placed central venous presure catheters not reaching the heart have a role. Pulmonary wedge pressure measurements should generally be deferred until

after rewarming.[130] The need for red cell transfusions is determined by the corrected hematocrit. Dilution with warmed infusate does not cause significant hemolysis.[165]

In some cases, rapid volume expansion is critical.[22,96,146] The circulatory volume is decreased, and peripheral vascular resistance increased.[194] In neonates, adequate fluid resuscitation markedly decreases mortality.[502] Adults receiving hemodynamic and pulmonary wedge pressure monitoring have shown improvement of cardiovascular efficiency during crystalloid administration.[194]

REWARMING

Passive External Rewarming

Since no controlled studies exist, rigid treatment protocols should not be suggested.[560,73] A versatile approach to rewarming considers results from animal experiments, human experiments on mild hypothermia, and clinical reports.[505,439,212,463,97]

The initial treatment decision is to determine the necessity of active versus passive rewarming. Noninvasive passive external rewarming (PER) is ideal for the majority of previously healthy patients with mild hypothermia. The patient is covered with dry insulating materials in a warm environment to minimize normal mechanisms of heat loss. When wind is blocked, less heat escapes via radiation, convection, and conduction. Conditions with higher ambient humidities limit respiratory heat loss.

Endogenous thermogenesis must generate an acceptable rate of rewarming for PER to be effective. Humans are poikilothermic below 30°C. and metabolic heat production is less than 50% of normal below 28°C.[35] Shivering thermogenesis is extinguished below 32°C. This thermoregulatory

neuromuscular response to cold normally increases heat production from 250 to 1000 Kcal/hr unless glycogen is depleted during cooling.[402]

Elderly patients who gradually develop mild hypothermia are good candidates for PER. Peripheral vasoconstriction is maintained, which minimizes core temperature afterdrop. If rewarming times are prolonged over 12 hours, complications increase. Clinical experience with PER is summarized in Table 2.

Previous recommendations for rewarming rates with PER varied between 0.5°C and 2.0°C/hr.[303,93] Patients who are centrally hypovolemic, glycogen depleted, and without normal cardiovascular responses should be stabilized and rewarmed at a conservative rate.[85,126] In a recent multicenter survey, the first (0.75 ± 1.16), second (1.17 ± 1.17), and third (1.26 ± 1.28) hour rewarming rates for the elderly far exceeded 0.5°C with no increase in mortality.[97]

ACTIVE EXTERNAL REWARMING

Active rewarming, the direct transfer of exogenous heat to a patient, is usually required below 32°C. Rapid identification of any impediments to normal thermoregulation is essential. Common denominators are cardiovascular instability and endocrinologic insufficiency.[329,303,423,22,146,164,41,272,72] Thermogenesis may also be insufficient following traumatic spinal cord transection or pharmacologically induced peripheral vasodilatation.[18,397] Aggressive rewarming of infants minimizes energy expenditures and decreases mortality. Vigorous monitoring for respiratory, hematologic, metabolic, and infectious complications is essential.[561,502,238,483,407]

When active rewarming is needed, heat can be delivered externally or to the core. Active external rewarming (AER) techniques deliver heat

directly to the skin.[147,273,327] Plumbed garments, hot water bottles, heating pads and blankets, and radiant heat sources have been used.[423,196,272] Thermal injury to poorly perfused vasoconstricted skin with these external heat application techniques is a hazard in adults and in children.[89,141,79] Immersion in a 40°C circulating bath is another option. Monitoring, resuscitation, treatment of injuries, and maintenance of extremity vasoconstriction to prevent core temperature afterdrop is difficult. CPR will prove impossible.

Initial concern with AER was expressed by Duguid in 1961, after 20/23 of her patients died.[119] Retrospective analysis of numerous clinical series noted a disproportionately high mortality rate with AER (Table 3).[183] In one study, the mortality rate was 64.3%.[329]

Interpretation of survival rates with AER is affected by risk factors and patient selection criteria. In some reports, "active" is an artificial description since rewarming required over 24 hours.[147] One author reported a 100% success rate with immersion rewarming.[162] DeRoubaix successfully reanimated a 13-year-old boy in cardiopulmonary arrest at 25°C in a hut turning him by the glow of a fire in a manner "similar to spit roasting".[105]

Experimental and clinical reports have linked AER with peripheral vasodilatation, hypotension, and core temperature afterdrop.[329,303,183,348,333] In one canine model, three groups were treated with partial cardiac bypass, peritoneal dialysis, and AER with a circulating blanket.[346] The externally rewarmed group required more bicarbonate and two to three times the crystalloid volume to maintain cardiac filling pressures as the other two groups. Sudden periodic acidotic shifts in arterial pH were seen even when the temperature exceeded 30°C.

These pH fluctuations were accompanied by conversion from sinus rhythm to VF in some animals. Isolated AER is never the rewarming technique of choice.

While previously healthy, young, and acutely hypothermic victims are usually safe candidates for AER, it offers no advantages.[97,174] If selected, heat application should be confined to the thorax; otherwise the depressed cardiovascular and metabolic systems may not meet peripheral demands.[196,197] Combining truncal AER with active core rewarming may avert many of this treatment's side effects.[272,511] AER by immersion is not recommended.

ACTIVE CORE REWARMING

A variety of techniques effectively deliver heat to the core. Options include heated inhalation, IV fluids, gastro-intestinal irrigation, peritoneal dialysis, extra-corporeal rewarming, and diathermy.[183,561,84,551,369] (Table 4) Average first hour rewarming rates reported with these techniques in a multicenter study are listed in Figure 9.

Airway Rewarming

Heated humidified oxygen inhalation has been studied extensively for prehospital and ED rewarming.[329,84,458,459,460,294,295,274,293,202,412] The effectiveness of the respiratory tract as a heat exchanger varies with technique and ambient conditions.[339,536] Since dry air has very low thermal conductivity, complete humidification and an inhalant temperature of 40°-45°C is required. Heat yield represents 10% to 30% of the hypothermic patient's heat production when the respiratory minute volume is adequate.[202,292,186] Heat transfer is greater through an endotracheal tube than by mask.[97,290,331]

The quantity of heat liberated during airway rewarming is mainly produced from condensation of water vapor. The latent heat of vaporization of water in the lung is slightly lower than 540 Kcal/g of H_2O at 100°C. This is multiplied times the liters per minute ventilation to calculate heat transfer. When the core temperature is 28°C, the rate of rewarming with heated ventilation at 42°C equals endogenous heat production.[354]

Heated humidified inhalation assures adequate oxygenation, stimulates pulmonary cilia, and reduces the amount and viscosity of the cold-induced bronchorrhea.[343,344,283] Although pre-existent PVC's may reappear during rewarming, there is no evidence that inhalation rewarming precipitates new clinically significant ventricular arrhythmias.[97,329] Vapor absorption does not increase pulmonary congestion or wash out surfactant.[513] While the pulmonary vasculature is heated, warmed oxygenated blood returning to the myocardium attenuates intermittent temperature gradients.[344,27] The amplitude of shivering is lowered, which is an advantage in more severe cases. This suppression can decrease heat production in mild hypothermia, although experimentally the core temperature rises.[344]

There are numerous oxygenation considerations in hypothermia[251,28,342,] (Figure 10). The "functional" value of hemoglobin has been calculated at 28°C to be 4.2 g/10 g in patients on cardiopulmonary bypass.[149] The oxyhemoglobin dissociation curve shifts to the left (Figure 4). In canine experiments, no evidence was found that this left shift impairs oxygen extraction by tissues during hypothermia.[187] The hypothermic dogs tolerated hypoxemia better than normothermic controls.

Although some patients self-adjust their RMV for current CO_2 production, there may be additional toxicologic or metabolic

depressants.[455,459] A cascade nebulizer with an immersion heater is adequate equipment for patients with spontaneous respirations. Ideally, the inhalation hose has a surrounding warming wire. An in-line disposable temperature monitor is necessary.[473]

Without modification, many commercially available heated nebulizers do not allow the temperature to reach the desired 40-45°C. All modified equipment should be labeled to avoid routine use.[473,458] A volume ventilator with a heated cascade humidifier can also deliver CPAP or PEEP if needed during rewarming. The airway rewarming rates clinically range between 1-2.5°C/hour.[329,84,459,97,295,343]

Although airway rewarming provides less heat than some other forms of active core warming, it prevents heat and moisture loss normally occurring through respiration. It is a safe, noninvasive, practical technique in all settings.

Heated humidified oxygen via facemask may not be possible in some patients with coexistent facial trauma. Airway rewarming is indicated in the field when equipment is available, and in virtually all cases $\leq$ 32.2°C on arrival at the emergency department.

Heated IV's

Intravenous fluids should be heated to 40 - 42°C. The amount of heat provided by solutions becomes significant in massive volume resuscitations. One liter of fluid at 42°C will provide 14 KCal to a 70 Kg patient at 28°C, elevating the core temperature almost 1/3°C.[354] Fluid resuscitation of hypovolemic patients can induce hypothermia.[462] In one series of previously normothermic patients with major abdominal vascular trauma, the average post resuscitation temperature was 31.2°C in those with refractory coagulopathies.[239]

IV fluids in flexible plastic containers can be heated with thermal packs or in a microwave oven more rapidly than with most standard blood warmers.[535,14] The plasticizer in the polyvinyl chloride containers is stable to microwave heating. Complete screening of other potentially leachable phthalate compounds has not been done. Heating times should be determined for each individual microwave oven, and average around 2 minutes at high power for a liter bag of crystalloid. The fluid should be thoroughly mixed prior to administration, since "hot spots" are common in most ovens. Fresh frozen plasma can be thawed in under five minutes. Infuse fluids through short tubing. Central administration of fluids at a temperature significantly different than the blood into the right atrium may produce myocardial thermal gradients.[348,177,556] Blood pre-heated in a standard warmer is useful,[40] but clotting and shortened red cell life is a hazard with blood warming packs.[34] Local microwave over-heating will hemolyze blood. An alternative is to dilute packed red blood cells with warm calcium-free crystalloid.[271] The Level 1 Fluid Warmer (Life Systems, Inc., Southfield, MI) warms cold crystalloids and blood from 10°C to 35°C via a heat exchanger at flow rates to 500 ml/min. Intravenous solutions and blood should be routinely heated during all hypothermic resuscitations.

Heated Irrigation

Heat transfer from irrigation fluids is usually limited, and should not be used as the sole rewarming technique. Direct gastrointestinal irrigation is less desirable than via intragastric or intracolonic balloons because of the induced fluid and electrolyte fluxes. A double-lumen esophageal tube has been investigated, as have other modified Sengstaken tubes.[259]

In direct gastric lavage, warmed electrolyte solutions like normal saline or Ringer's are administered via nasogastric tube. After 15 minutes, the solution is aspirated and replaced with warm fluids. Disadvantages include the small surface area available for heat exchange, and the large amount of fluids escaping into the duodenum. Regurgitation is common, and the technique must be terminated during CPR.

Mediastinal irrigation and direct myocardial lavage are alternatives in patients without spontaneous perfusion.[154,52,329,41,423,183,22,47,303,196,272,284,87] A standard left thoracotomy is performed while CPR is continued. Do not open the pericardium unless an effusion or tamponade is present. Bathe the heart for several minutes in one to two liters of an isotonic electrolyte solution heated to 40°C. This is followed by suctioning and replacement of warm fluids.

Attempt internal defibrillation at 1 - 2°C intervals after the myocardial temperature reaches 26 - 28°C. When a perfusing rhythm is achieved, continue lavage until the myocardial temperature exceeds 32° - 33°C. As suggested by O'Keeffe, the standard post-thoracotomy left chest tube could provide an avenue for continued rewarming via irrigation.[377]

Bilateral afferent and efferent thoracostomy tube irrigation has been evaluated on dogs.[52] Continuous warm saline thoracic cavity lavage was more effective than gastric lavage. Do not perform this technique on patients with spontaneous perfusion, since chest tube insertion may directly precipitate ventricular fibrillation.

Unless immediate cardiopulmonary bypass is an option, mediastinal irrigation and direct myocardial lavage are indicated only in arrested

patients. In these circumstances, personnel skilled in the technique should combine it with all available rewarming modalities.

Peritoneal Lavage/Dialysis

Heated peritoneal lavage is a technique available in most facilities.[478] Heat is conducted intraperitoneally via isotonic dialysate delivered at 40 - 45°C.[253,269,424] This technique is not a practical field option.

Before lavaging, obtain chest and abdominal radiographs, since subsequent films may reveal subdiaphragmatic air introduced during the procedure. The bladder and stomach must be emptied prior to inserting the catheter. There are two popular variations of the technique for introducing fluid into the peritoneal cavity: the "mini-lap" and the percutaneous puncture.

The mini-lap requires an infraumbilical incision through the linea alba. Go supraumbilical if previous surgical scars, a gravid uterus, or pelvic trauma are identified. The peritoneum is punctured under direct visualization and the dialysis catheter inserted.

A much simpler and more rapid technique is the guide wire, or Lazarus-Nelson, variation of the percutaneous puncture.[377] A small diameter 18 gauge needle penetrates the peritoneum.
← The lavage catheter is twisted over the wire and advanced into one of the pelvic gutters. Double catheter systems with outflow suction speed rewarming. Isotonic dialysate is heated by an external bath to 40 - 45°C. Infuse around two liters, which is retained for 20 - 30 minutes and then aspirated. The usual clinical exchange rate is six liters/hour, which yields rewarming rates of 1 - 2°C per hour.[225,543,185,228,48,106,394]

A unique advantage of peritoneal dialysis is overdose and rhabdomyolysis detoxification when hemodialysis is unavailable. Additionally, direct hepatic rewarming reactivates detoxification and conversion enzymes. Peritoneal dialysis will worsen pre-existent hypokalemia. Vigilant electrolyte monitoring is essential prior to modification of dialysate.

Adhesions from previous abdominal surgery increase the complication rate and minimize heat exchange. Only one-third of the nonrenal dialyses were free of signifcant complications in one clinical report.[521] One of O'Connor's three stable severely hypothermic patients developed VF during the first exchange.[373,374]

Peritoneal dialysis during standard mechanical CPR was as effective as partial cardiac bypass in resuscitating severely hypothermic dogs.[346] Unlike the group of dogs receiving active external rewarming, significantly greater quantities of crystalloids and bicarbonate were not required during rewarming. In another canine study, peritoneal dialysis at a rate of 12 liters/hour rewarmed dogs more rapidly than heated humidified inhalation.[541] However, this exchange rate is rarely plausible in humans.

Peritoneal lavage should not be routinely used in treating stable hypothermic patients. It is invasive, and extracorporeal rewarming must be available in the event of major complications including ventricular fibrillation. In severe cases the transfer of heat is lower than that achieved with cardiac bypass or hemodialysis.[196,102,388] This technique should be used in combination with all available rewarming techniques in cardiac arrest patients.

Extra-corporeal rewarming

Cardiopulmonary bypass (CPB) and hemodialysis are life-saving modalities in selected profound cases of hypothermia. Althaus describes complete recovery in three severely refrigerated tourists after prolonged periods of cardiac arrest and CPR (Table 5).[5] In another review of 17 cases, there were 13 survivors.[481]

A major advantage of CPB is the preservation of flow if mechanical cardiac activity is lost during rewarming.[423,144,101,547,275,514,5,113,262,402,60,510] CPB should also be considered when severe cases do not respond to less invasive rewarming techniques, in patients with completely frozen extremities, and when rhabdomyolysis is accompanied by major electrolyte disturbances.

CPB should only be performed by an experienced team. After the groin cutdown is completed, the femoral artery and vein in adults or iliac vessels in small children are cannulated. Intravenous anticoagulation with heparin is required.

The warmer is set at 38 - 40°C, and heated oxygenated blood is returned via femoral artery. Femoral flow rates of 2 - 3 liters per minute can elevate core temperature 1-2°C every 3 - 5 minutes. In Splittgerber's review, the mean CPB temperature increase was 9.5°C per hour.[481] Full flow rates up to 6 liters per minute can be achieved with the portable Bard CPS® system.

Hemodialysis will become a more widely available and practical rewarming technique with the development of two-way flow catheters allowing cannulation of a single vessel.[377] A Drake-Willock single-needle

dialysis Controller can be used with a portable hemodialysis machine and external warmer. After central venous cannulation, exchange cycle volumes of 200-250 ml per minute are possible. Although heat exchange is less than standard two-vessel hemodialysis with CPB,[354] the ease of percutaneous subclavian vein placement is a major advantage. Local vascular complications including thrombosis of vessels and hemorrhage secondary to anticoagulation may occur.[453,517]

There is no proof that rapid acceleration of the rate of rewarming improves survival rates. Potential complications of uncontrolled rapid rewarming in severe hypothermia include DIC, pulmonary edema, hemolysis, and acute tubular necrosis. Nifedipine blunted cold-induced left ventricular dysfunction in a group of patients with systemic sclerosis.[132] Low dose IV vasodilator therapy with nitroglycerin might be a potential aid with rapid rewarming.

Rewarming should be attempted via CPB and hemodialysis in hypothermic cardiac arrest when CPR is not contraindicated (see CPR section) unless frozen intravascular contents are present, preventing flow. If experienced personnel and necessary equipment are unavailable, all other rewarming techniques should be used in combination.

Diathermy

Diathermy, the transmission of heat by convesion of energy, is being evaluated as a rewarming adjunct in accidental hypothermia.[492,445,196,379] Large amounts of heat can be delivered to deep tissues with ultrasonic (0.8 to 1.0 M Hz) and low-frequency (915 to 2450 M Hz) microwave radiation. Short-wave (13.56 to 40.68 M Hz) modalities are high frequency and do not penetrate deeply.[196] Contraindications include frostbite, burns, significant edema, and all types of metallic implants and pacemakers.

Under ideal conditions in a laboratory study, radio wave frequency (13.56 MHz) electromagnetic regional heating of hypothermic dogs after immersion did not damage tissue at 4-6 watts/kg and rapidly elevated the core temperature.[542] Zhong successfully rewarmed 16 piglets with microwave irradiation until they squealed and suckled. Subsequently, 20 of 28 human infants who were rewarmed with microwave irradiation at 90 to 100 watts survived.[561] The temperature rose an average 1°C after 6 - 7 minutes, and the average infant required 45 minutes to achieve a rectal temperature of 36°C.

Ultrasonic and low-frequency microwave diathermy have the potential to deliver large quantities of heat below the skin. As dosimetry guidelines are developed, potential complications and ideal application sites for this experimental technique deserve further study.

CPR

Basic and advanced life support recommendations in hypothermia continue to evolve.[97,487,503,484] Cardiac output generated with closed

chest compressions maintains viability in selected hypothermic patients. The optimal rate and technique is unknown.[75,518,260,63,479,272,87,444,365,94,514,10]

Definitive pre-hospital determination of cardiac activity requires the aid of a cardiac monitor. Misdiagnosis of cardiac arrest is a hazard.[63,64] Peripheral pulses are difficult to palpate when extreme bradycardia is combined with peripheral vasoconstriction.

Some authors contend that asystole is a more common presenting rhythm than ventricular fibrillation (VF).[22] In the field, differentiating VF from asystole may be impractical.[479,540,97,359] Potential etiologies of VF include acid-base fluxes, hypoxia, and coronary vasoconstriction with increased blood viscosity.[523,537,495,348,291,496] Chest compressions and various therapeutic interventions have been implicated.[22] The role of acid-base fluxes is not clear. Alkalosis appears protective against VF during controlled induced hypothermia.[260] Southwick has suggested alkalosis correlates with VF versus asystole and mixed acidosis.[479]

During normothermic conditions, blood flow results from phasic alterations in the intrathoracic pressure and not direct cardiac compression.[363,364,435] Niemann demonstrated antegrade flow occurs without left ventricular compression in a normothermic canine model.[363] Closed chest compression increases intrathoracic pressure.[534] When thoracic inlet venous valves are competent, the pressure gradient between the arterial and venous compartments causes supradiaphragmatic antegrade flow.[527]

During hypothermic cardiac arrest in swine, the cardiac output, cerebral, and myocardial blood flows averaged 50%, 55%, and 31% of that achieved during normothermic closed chest compressions.[310] Blood flow to these areas did not decrease over time, unlike in the normothermic group. There was no significant difference in flow generated between normothermic and hypothermic swine at 20 minutes. In hypothermia, the role of a "thoracic pump" with the heart as a passive conduit is an attractive hypothesis. There are a large number of neurologically intact survivors after prolonged closed chest compressions[5] (Table 5).

Chest wall elasticity is decreased with cold, as is pulmonary compliance.[524,104] More force is needed to depress the chest wall sufficiently to generate intrathoracic vascular compartment pressure gradients. Intrathoracic pressures have not been measured during hypothermic closed chest compressions. Pneumatic powered thoracic compression devices could be useful during prolonged resuscitations with limited availability of personnel.

Myocardial compliance can also be severely reduced in hypothermia. Althaus noted in 1 of 3 survivors at thoracotomy that "the heart was found to be hard as stone and it is hardly conceivable how effective external cardiac massage could have been".[5]

Hypothermic rheologic changes including increased viscosity also affect flow.[300,328] Peripheral vascular resistance would be expected to increase during vasoconstriction.[552,559] However in swine, there was no difference in systemic and organ vascular resistance between normothermic and hypothermic CPR.[310]

In a multicenter survey of 428 cases, nine of 27 patients receiving CPR initiated in the field survived, as did 6 of 14 patients with ED initiated CPR.[97] Based on these cases and a literature review (Table 5), the following refinements of the American Heart Association's CPR standards in hypothermia have been proposed.[97,484]

Initiate CPR in accidental hypothermia unless: *

1.) Do not resuscitate status is documented and verified.

2.) Obviously lethal injuries are present.

3.) Chest wall depression is impossible.

4.) Any signs of life are present.

5.) Rescuers are endangered by evacuation delays or altered triage conditions.

Tissue decomposition, apparent rigor mortis, dependent lividity, and fixed dilated pupils are not reliable criteria for withholding CPR.[74,484] Intermittent flow may provide adequate support during evacuation.[336,188,215,334] One should not withhold CPR only because continuous compressions cannot be assured.[400,24,372,392] The lowest temperature documented in an infant survivor of accidental hypothermia is 15.2°C, in an adult is 16°C, and in induced hypothermia is 9°C.[368,99,360]

When cardiopulmonary arrest develops during resuscitation, other causes include pulmonary emboli and respiratory insufficiency.[104] An adequate oxygen supply is essential during rapid rewarming.[454,515,67,209] For each 10°C rise in temperature, oxygen consumption increases 2.5 - 3 times.[35,149]

* Developed in conjunction with the Wilderness Medical Society

Endotracheal intubation and ventilation decrease atelectasis and ventilation-perfusion mismatch.[95] Complete airway protection averts aspiration which is otherwise common in the setting of hypothermic airway reflex depression, bronchorrhea, and ileus.[398] Carbon dioxide production halves with an 8°C fall in temperature. Ventilation should be adjusted accordingly. During induced hypothermia, carbogen (1 -5% CO_2 added to oxygen) facilitated acid-base management by allowing adjustment of the F_ICO_2.[303,454,529,191,319]

Controversy regarding the hazards of endotracheal intubation reflects coincidental episodes and a miscitation by Fell of a series of hypothermic overdoses by Lee & Ames.[144,275] Fell stated that "endotracheal intubation was followed by cardiac arrest in a large proportion of cases", while Lee & Ames merely cautioned of that possibility. In a multicenter survey, endotracheal intubation was performed on 117 patients by multiple operators in various settings.[97] No induced arrhythmias were recognized, which is consistent with several reports.[511] Danzl and Miller nasotracheally intubated 40 hypothermic patients without incident,[95,329] Ledingham noted no arrhythmias in his prospective series of 44 cases. Potential arrhythmogenic factors include hypoxia, mechanical jostling, and acid-base or electrolyte fluctuations.

Indications for endotracheal intubation in hypothermia are identical to those in normothermia.[172,96,63] It is required unless the patient possesses intact protective airway reflexes. Ciliary activity is depressed in hypothermia, frothy sputum produces chest congestion, and this bronchorrhea resembles pulmonary edema.[195,196] Blind nasotracheal

intubation is preferable to cricothyroidotomy when cold-induced trismus or potential cervical spine trauma is present.

Resuscitation Pharmacology

Pharmacologic effects of medications are temperature dependent. The lower the temperature, the greater the protein binding. Entero-hepatic circulation and renal excretion are altered and abnormal physiologic drug responses should be anticipated. The usual clinical scenario is substandard therapeutic activity while the patient is severely hypothermic progressing to toxicity after rewarming. Oral medications are contraindicated because of decreased gastrointestinal function. Intramuscular medications may be erratically absorbed from vasoconstricted sites.[303]

Pharmacologic manipulation of respiratory drive, pulse, and blood pressure is generally not indicated. When relative tachycardia is not consistent with temperature depression, one should consider hypovolemia, hypoglycemia, and toxicologic etiologies.[86] Vasopressors are arrhythmogenic and cannot increase peripheral vascular resistance if the vasculature is maximally vasoconstricted.[303,272] Vasodilators can precipitate core temperature afterdrop. If intra-arterial pressure is not consistent with the degree of hypothermia, judicious use of inotropic agents may be necessary. In one clinical report, dopamine was a successful adjunctive treatment. Two patients with profound hypothermia following ethanol ingestion were resuscitated with low-dose dopamine support.[408] In frostbite victims, however, the decision to use catecholamines may jeopardize extremities. Catecholamines will also exacerbate pre-existent occult hypokalemia.

The effects of temperature depression on the autonomic nervous system are being investigated. The sympathetic nervous system in primates has a

biphasic response in plasma catecholamine concentrations in response to cooling. After an initial increase, it switches off at 29°C.[71] This suggests catecholamine support might be useful below that temperature. The initial rise in catecholamine levels could also be caused by acute respiratory acidosis stimulating the sympathetic nervous system.[46]

Dopamine alone or with lidocaine in another animal study improved cardiovascular function equivalent to a 5°C rise in temperature.[361] In one clinical series, endogenous catecholamine levels were elevated during bradycardia. As the levels dropped during rewarming, the pulse rate increased.[190] Low dose dopamine (1 to 5 μg/kg/min) infusions should be reserved for severely hypotensive patients not responding to crystalloid resuscitation and rewarming.

Atrial Arrhythmias

All atrial arrhythmias, including atrial fibrillation, should have a slow ventricular response with temperature depression. Atrial fibrillation was commonly noted below 32°C in an analysis of 60 EKG's from accidental hypothermia victims.[375] In half the cases, the rhythm was sinus, atrial, or junctional. In two other series, atrial fibrillation was reported in 12/102 and 12/33 cases.[539,376]

Atrial fibrillation usually converts spontaneously during rewarming, and digitalization is not warranted.[30,355,500] The AH interval prolongation present on HIS bundle electrocardiography is unresponsive to atropine.[223] Mesenteric embolization is a potential hazard when the rhythm converts back to sinus.[93]

Hypothermia renders the negative inotropic effects of calcium channel blockers redundant.[207] Verapamil has been used in the resuscitation of a profoundly hypothermic near-drowning case.[257] Any additional cerebral protective effects are speculative.

All new atrial arrhythmias will usually convert spontaneously during rewarming, and should be considered innocent. Correct acid-base, fluid, and electrolyte imbalances. Administration of atrial anti-arrhythmics is not indicated.

Ventricular Arrhythmias

The role of prevention and the ideal treatment of ventricular arrhythmias in hypothermia are not resolved. Since pre-existent chronic ventricular ectopics may be suppressed in a cold heart, the physician noting these ectopics during rewarming is placed in a quandry. Past cardiac history is often unavailable.[316]

Transient ventricular arrhythmias should be ignored. In a study of 22 continuously monitored hypothermic patients, supraventricular arrhythmias were common (9 cases) and benign.[417] Ten patients developed ventricular extrasystoles but none developed VT or VF during rewarming. The terminal rhythm in the 8 who died while being monitored was asystole, not VF. The incidence of iatrogenic VF is low in accidental hypothermia if patients are well oxygenated and handled carefully.

Pharmacologic options are limited since hypothermia induces complex physiologic changes which result in abnormal responses.[550] Drug metabolism and excretion are both progressively decreased. In normothermia, group 1 ventricular antiarrhythmics have indirect anticholinergic effects and decrease conduction velocity.[523] Procainamide reportedly increases the incidence of VF. Quinidine has been useful during induced profound hypothermia,[121] preventing VF during cardiac manipulation at

25°C-30°C. Magnesium sulfate at a dose of 100 mg/Kg IV spontaneously defibrillated most CPB patients at 30°C in one series of patients with induced hypothermia.[57] This occurred within minutes in two-thirds of cases. Lidocaine has not been effective for prophylaxis, and is ineffective in facilitating defibrillation.[362,13] In animal studies, lidocaine and propranolol have minimal hemodynamic effects in hypothermia.[361]

Bretylium tosylate, a unique bromobenzyl quarternary ammonium compound with hypothermic activity, has been extremely effective in several animal studies.[362,13,55,254,116,98] Nielson found bretylium increased the fibrillation threshold in cats, and Buckley demonstrated efficacy in the prophylaxis and treatment of VF in a canine model.[362,55] While 42% of controls fibrillated, no dog given 15 mg/Kg bretylium did so. In the design of both of these studies, the drug was administered prior to inducing hypothermia. In a similarly designed canine study by Elenbaas, bretylium to facilitate defibrillation at 22°C.[131]

In the first study to evaluate the effects of bretylium administered after induction of hypothermia, Murphy noted that only one of eleven dogs given 5 mg/Kg bretylium prior to five invasive maneuvers developed VF.[350] No dog, including controls, fibrillated during endotracheal intubation. Of note in considering prohylaxis, 3 of the 11 dogs converted to VF during the drug infusion.

Emergency transvenous intracardiac pacing of bradyarrhythmias is extremely risky because it commonly precipitates ventricular fibrillation. New dysrhythmias that develop after rewarming may require pacing on rare occasions. External noninvasive pacing with low resistance electrodes seems preferable prior to stabilization.[385,347,139,138]

In summary, although human data are sparse, bretylium is the only agent shown to have anti-arrhythmic activity during hypothermic conditions. Two cases of chemical defibrillation after infusion of 10 mg/Kg bretylium in accidental hypothermia have been reported.[94,255] Bretylium prophylaxis is investigational, since toxicity, optimal dosage and particularly the ideal rate of infusion are not known. Bretylium appears to be the agent of choice for VF in hypothermia.

Thyroid

The indications for administering thyroid hormone in hypothermia are controversial, as are the optimal dose and preparation.[123,155,549,426,519,451,21,218] Cold induces stimulation of the hypothalamic-pituitary-thyroid axis. Unless myxedema is suspected, empiric therapy is no longer recommended.[303,423,329,539,272,103] A history of neck irradiation, radioiodine or surgical treatment of hyperthyroidism, or Hashimoto's thyroiditis should heighten suspicion for myxedema. Failure to rewarm despite an appropriate course of therapy is a further clue.

Myxedema coma is usually precipitated in elderly patients with chronic hypothyroidism who are stressed by trauma, infection, anesthesia, or medication ingestion. Typical non-specific laboratory abnormalities include hyponatremia, anemia, liver enzyme and lipid elevations.

If myxedema coma is suspected, obtain thyroid function studies including serum thyroxine (T_4) by radioimmunoassay, triiodothyronine (T_3) resin uptake,, and thyroid-stimulating hormone. Also, measure the serum cortisol level.

Administer 250 - 500 µg levothyroxine (T_4) intravenously over 30 - 60 seconds without wating for confirmatory laboratory results. Daily

injections of 100 µg are required for 5 - 7 days.[103,426,519,451,21,218] Add 100 to 200 mg of hydrocortisone to each liter of intravenous fluid. Absorption of levothyroxine is erratic if given orally or intramuscularly. The onset of action of T_3 is more rapid, which jeopardizes cardiovascular stability.[218] There is no current role for T_3 in acute replacement therapy.[103,1]

Identify and treat precipitating causes of hypothermic myxedema coma. Active external rewarming is contraindicated because it causes vasodilation and vascular collapse. The risk of delayed treatment of a patient in myxedema coma exceeds the risk of giving T_4 to a euthyroid hypothermic patient.

Steroids

Acute cold stress stimulates cortisol secretion, as do many coexistant disease processes. The free active fraction of cortisol decreases with temperature depression because of increased protein binding. Cortisol utilization is similarly decreased.[378]

The increase in ACTH and adrenal steroid secretion may also be a neurogenic or emotional response in the conscious subject to an unpleasant environment. In rodents, inhibition of ACTH secretion during hypothermia is mediated by decreased hypothalamic secretion of arginine vasopressin and oxytocin. This decreases pituitary responsiveness to corticotropin-releasing factor, inhibiting corticotropin release. Thus exogenous arginine vasopressin could prove helpful during rewarming.171

Canine experiments have demonstrated potential cerebral protective effects of 4 mg/kg dexamethasone IV in cold-injured cortical microcirculation.[480] In a clinical report, intracranial pressure remained

normal without steroid supplementation during rewarming from 23°C.[370,19]

Cold exposure also induces adrenal unresponsiveness to adrenocorticotropic hormone. False diagnosis of a decreased adrenal reserve is possible.[142,143] This does not represent functional adrenal insufficiency, since ACTH returns to normal after rewarming. Serum cortisol levels are commonly elevated.[329,272] Secondary adrenal insufficiency from panhypopituitarism may coexist with myxedema.[303] Steroids should be withheld unless hypoadrenocorticism is proven or is suspected because of previous history of steroid dependence.[329,539,549,378] If the patient fails to rewarm, reassess the situation.

Use of narcotic antagonists in hypothermia has been reported. Naloxone has been implicated in reducing the severity of hypothermia in overdoses and in spinal shock.[217,173] Currently, the indication for its use should remain antagonism of opiate receptor sites.

Reported treatments of thromboembolism in hypothermia are sparse. Continue active rewarming, infuse fresh frozen plasma, and empirically treat for sepsis. Heparin should be reserved for documented thromboembolic disease.[423,65]

Sepsis

The pathophysiology of sepsis in hypothermia continues to be investigated.[32] Classical signs of infection, including erythema and fever, are absent. Rigors resemble shivering. The initial history,

physical, and laboratory data are often unreliable, and therefore repeated evaluations and comprehensive culturing are mandatory in the Emergency Department (ED).[401]

Host defenses are compromised and serious bacterial infections are common. Significant infections can be accompanied by a minimal inflammatory response.[279] Bone marrow release and circulation of neutrophils is compromised for up to 12 hours.[31] Human and porcine neutrophils are susceptible to hypothermia.[494] In vitro, neutrophil migration and bacterial phagocytosis are reduced at 29°C.[2] Neutrophil killing of Staphylococcus aureus and Streptococcus faecalis is impaired.

Therapeutic maintenance of hypothermia in regimens to control cerebral edema in near-drowning is being abandoned because of the substantial incidence of infectious complications.[37] Acquired neutrophil dysfunction has been identified.[76] In addition, hypothermia was associated with decreased number of neutrophils in a series of 40 near-drowned children.[37] (See Near-drowning Chapter)

The reported incidence of infection varies dramatically age and series.[539,279,110] In one group of 51 infants, 27 were septic.[92] While there were no reliable indicators of infection, some suggestive clues were present. Serum glucose and leukocyte abnormalities, anemia, uremia, and bradycardia were often identified. In addition to Staphylococcus and Streptococcus, predominant organisms were Hemophilus and Enterobacteriaceae. Nine of Yagupsky's 57 hypothermic infants were septic.[554,555]

Lung infections, usually in the right upper lobe, were reported in 80 of 138 hypothermic infants by El-Radhi.[133,134] Gastric aspirate was another diagnostic aid for sepsis in 36 of 44 infected infants.[135] In five studies, from 8% to 74% of hypothermic infants were septic.[92,133-135,438] In this age group, empiric broad-spectrum antibiotics are warranted. DAgan recommends an intravenous aminoglycoside with ampicillin.[92]

In adults, the incidence of infections in hypothermia ranges from less than 1% to over 40% depending on patient selection criteria.[279,539,340] In Lewin's series, serious soft tissue or pulmonary infections were present in 24 of 59 patients.[279] Nine were undiagnosed at hospital admission from the ED. Occult bacteremia was present in less than 1% of White's series of 102 patients and none of the 46 lumbar punctures were positive.[539]

Infection with bacteremia was identified in 33 of 85 consecutive cases by Morris.[340] In this series, 32 patients were hemodynamically monitored. Prior to rewarming, the combination of elevated cardiac index and decreased systemic vascular resistance suggested bacteremia. Although there were no reported complications in this series associated with right heart catheterization, this coincidental observation should prompt a search for infection when hemodynamic monitoring is indicated for other reasons.[401,340]

In summary, unlike in children, empiric antibiotic prophylaxis is not warranted for most adults. However, treatment indications should be liberalized from normothermia and include failure to rewarm or any suspicion or evidence of aspiration, myositis, chest x-ray infiltrates,

bacteriuria, or persistent altered mental status. Common infections include the gram negatives, gram positive cocci, oral anaerobes, and enterobacteriaceae.[279,340,221] Lewin cultured Escherichia coli, Streptococcus pneumoniae, two Proteus and Klebsiella species, and Staphylococcus aureus and epidermidis.[279] In choosing broad spectrum coverage, consider the altered drug interactions, volumes of distribution, protein binding, hepatic metabolism, and renal excretion. An aminoglycoside and third-generation cephalosporin is often appropriate.

Forensic Pathology

Anatomic lesions are variable and nonspecific in hypothermia and there is no single pathognomonic finding at autopsy. Establishing hypothermia as the primary cause of death requires an adequate history of exposure and absence of other lethal findings at necroscopy.

Macroscopic skin changes can suggest the diagnosis. Hyperemia of the dorsum of the hands and the knees is commonly found. Non-pathognomonic findings have been identified in the pancreas, lungs and heart.[311,213] Pancreatic findings included fat necrosis, aseptic pancreatitis, and hemorrhage.[311,119] Pulmonic changes consisted of intra-alveolar, interstitial, and intrabronchial hemorrhages. Hirvonea also identified microscopic degeneration of the myocardium.[213] In Coe's post-mortem series, an interesting anatomic observation in 45% of the hypothermic deaths was low weight of the lungs.[78]

Bray initially observed an increased vitreous glucose concentration in a group of plane crash victims immersed in icy waters.[45] This suggests eye chilling might inhibit anaerobic glycolysis. Similar inhibition of

glucose consumption and lactate production was observed in experiments on decapitated sheep heads.[44]

The eye, directly exposed to the environment, could be a chemical indicator of environmental and patient temperature at the time of demise. Vitreous humor chemistry profiles on 133 autopsied patients revealed that glucose concentration and total CO_2 content varied inversely with temperature, with values significantly higher in winter months.[43] Elevated vitreous glucose in a nondiabetic victim suggests hypothermia.[78] Total urinary catecholamine content, particularly epinephrine, was high in a group of known hypothermic casualties.[214]

Prevention Optimal function in a cold environment requires an understanding of the principles of heat conservation and loss.[51] To maintain core temperature in the narrow band needed for peak function in cold environments, adaptive behavioral responses are essential. Autonomic and endocrinologic mechanisms are supplemental.[469,124,179]

Excellent physical conditioning with adequate rest and nutrition are paramount. Hikers and skiiers should be accompanied by a partner, and wear effective thermal insulation. Wet inner garments must be changed promptly. Persons who exert, including long-distance skiiers, should alter garment clo values depending on current exertional heat production.[476] Dehydration must be avoided. Drinking from a cold stream is preferable to snow ingestion, since it requires 80 Kcal to convert 1 kg of ice at 0°C to 1 kg of water at 0°C.[406]

All areas with a large surface area-to-volume ratio should be insulated. The uncovered head can lose up to 70% of the body's total heat production. A "nightcap", not originally Kentucky bourbon, is a stocking

cap worn to bed. Synthetic insulating materials include Gore-Tex®,Thinsulate®, and taslanized nylon.

Flectalon, a web of aluminized polyvinyl chloride fibers, is a new insulating material.[216] To compare its insulating efficacy with other materials, the "critical" temperature was determined. This is defined as the lowest environmental temperature at which the core temperature can be maintained without increased oxygen consumption. Flectalon lowers the critical temperature more than Thinsulate®, and may prove useful as an efficient insulator to prevent and treat hypothermia. The army mnemonic "COLD", in reference to insulation with clothing,is: clean, open during exercise to avoid sweating, loose layers retain heat, and dry limits conductive heat losses.[469] For a complete discussion of preventive measures, refer to the chapter "Wilderness Survival".

The prevention of urban accidental hypothermia requires public education. For example, the optimal safe indoor temperature recommendation for the elderly has risen to 21.1°C.[83,404] Energy assistance and temporary sheltering are effective. Selective heating of sleeping quarters and electric blankets are economical measures.[234]

Outcome

Survival is difficult to predict because of the variability of human physiologic response to temperature depression. The type and severity of the underlying or precipitating disease process is one determinant. Age extremes, while not statistically correlated with survival, are commonly associated with severe illnesses.[329,538,539,272,219,376,270] In a multicenter survey, there were no significant differences by age in mortality.[97]

Sex, trauma, infection, and toxin ingestions impact survival differently in multiple uncontrolled clinical studies. There were no clinically significant differences in male versus female profiles in a multicenter survey.[97] A hypothermia outcome score is being developed by Hedges on a large hypothermia database which will enable multiple observers at differing sites to assess treatment modalities and outcome predictors. Prehospital cardiac arrest, a low or absent presenting blood pressure, elevated BUN, and the need for either endotracheal or nasogastric intubation in the emergency department were significant predictors of outcome after multivariant analysis.[97,272,329,539,314,376,538,558,232,150]

Summary

Hypothermia continues to successfully masquerade in a variety of settings. Field treatment should include gentle handling, insulation, and heated humidified oxygen. Active external rewarming should only be used selectively and be limited to the trunk. Individualized ED rewarming selections require a versatile approach based on the pathophysiology, presentation, and available facilities. Active core rewarming with heated humidified oxygen is a safe practical option in all ED's.

Patients with mild primary accidental hypothermia can be rewarmed in the ED and discharged. Those with serious predisposing conditions require hospital admission and intensive care. Moderate or severely hypothermic patients ($\leq 32.2^{\circ}C$) require hospital admission if predisposing conditions, age extremes, or abnormal laboratory or toxicologic values are present.

Transfer to specific centers specializing in a signle rewarming technique is not generally warranted. No survival advantage has been identified for any particular rewarming modality. Some severely hypothermic patients are best managed in facilities with cardiopulmonary bypass capabilities. Choice of a specific rewarming technique should reflect available expertise and resources.

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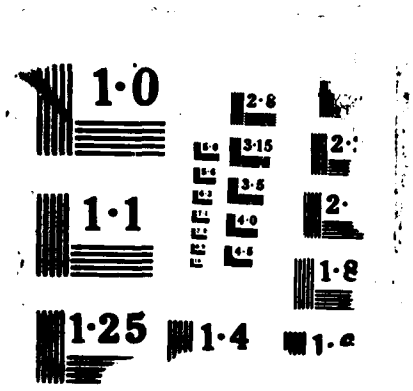
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